	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments	Error Definition	Errors
1	BRS	L1	55	pr-39	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/03/30 17:51			0
2	BRS	L3	3	pr-39 same oligopeptide	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/03/30 17:52			0
3	BRS	L4	465	proteasome	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/03/30 17:53			0
4	BRS	L5	210	4 same (inhibition or inhibitor)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/03/30 17:53			0
5	BRS	L6	2	1 same 5	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/03/30 17:53			0

> d his

(FILE 'HOME' ENTERED AT 17:56:41 ON 30 MAR 2002)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
17:57:05 ON 30 MAR 2002
L1 364 S PR-39
L2 21657 S PROTEASOME
L3 9946 S L2 (P) INHIBIT?
L5 9 S L1 (P) L3
L6 6 DUPLICATE REMOVE L5 (3 DUPLICATES REMOVED)

=> log y

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=> file medline caplus biosis embase scisearch agricola
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
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FULL ESTIMATED COST                                     0.21       0.21

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=> s pr-39
L1          364 PR-39

=> s proteasome
L2        21657 PROTEASOME

=> s l2 (p) inhibit?
L3         9946 L2 (P) INHIBIT?

=> s l1 (p) l1
L4          364 L1 (P) L1

=> s l1 9p) l3
MISSING OPERATOR L1 9P
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s l1 (p) l3
L5            9 L1 (P) L3

=> duplicate remove l5
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L5
L6             6 DUPLICATE REMOVE L5 (3 DUPLICATES REMOVED)

=> d l6 1-6 ibib abs

L6   ANSWER 1 OF 6  CAPLUS  COPYRIGHT 2002 ACS
ACCESSION NUMBER:         2001:489246  CAPLUS
DOCUMENT NUMBER:          135:87168
TITLE:                    Method for PR-39 peptide-mediated selective inhibition
                          of I.kappa.B.alpha. degradation
INVENTOR(S):              Simons, Michael; Gao, Youhe
PATENT ASSIGNEE(S):       Beth Israel Deaconess Medical Center, USA
SOURCE:                   PCT Int. Appl., 68 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     ---------------   ----  --------       ---------------  --------
     WO 2001047540      A1   20010705       WO 2000-US35293  20001227
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W: AU, CA, JP
RW: AT, BE, CH, CY, DE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR
PRIORITY APPLN. INFO.: US 1999-474967 A 19991229
AB The invention provides both a method and means for regulating I.kappa.B.alpha. degrdn., NF.kappa.B activity, and NF.kappa.B-dependent gene expression within living cells, tissues, and organs in-situ. The selective regulation is performed using native PR-39 peptide or one of its shorter-length homologs, for interaction with such I.kappa.B.alpha. and proteasomes as are present in the cytoplasm of viable cells. The result of PR-39 peptide interaction with I.kappa.B.alpha. is a selective alteration in the intracellular proteolytic activity of proteasomes, which in turn, causes a redn. of I.kappa.B.alpha., a decrease of NF.kappa.B activity, and a down-regulation of NF.kappa.B-dependent gene expression.

L6 ANSWER 2 OF 6 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2001663431 MEDLINE
DOCUMENT NUMBER: 21565666 PubMed ID: 11709430
TITLE: ***PR*** - ***39*** and PR-11 peptides ***inhibit*** ischemia-reperfusion injury by blocking ***proteasome*** -mediated I kappa B alpha degradation.
AUTHOR: Bao J; Sato K; Li M; Gao Y; Abid R; Aird W; Simons M; Post M J
CORPORATE SOURCE: Angiogenesis Research Center, Dartmouth Medical School, Hanover, New Hampshire 03756, USA.
CONTRACT NUMBER: HL-53793 (NHLBI)
HL-636-09 (NHLBI)
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY. HEART AND CIRCULATORY PHYSIOLOGY, (2001 Dec) 281 (6) H2612-8.
Journal code: 100901228. ISSN: 0363-6135.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200201
ENTRY DATE: Entered STN: 20011119
Last Updated on STN: 20020125
Entered Medline: 20020107
AB ***PR*** - ***39*** ***inhibits*** ***proteasome*** -mediated I kappa B alpha degradation and might protect against ischemia-reperfusion injury. We studied ***PR*** - ***39*** , its truncated form PR-11, and a mutant PR-11AAA, which lacks the ability to prevent I kappa B alpha degradation, in a rat heart ischemia-reperfusion model. After 30 min of ischemia and 24 h of reperfusion, cardiac function, infarct size, neutrophil infiltration, and myeloperoxidase activity were measured. Intramyocardial injection of 10 nmol/kg ***PR*** - ***39*** or PR-11 at the time of reperfusion reduced infarct size by 65% and 57%, respectively, which improved blood pressure, left ventricular systolic pressure, and relaxation and contractility (+/-dP/dt) compared with vehicle controls 24 h later. Neutrophil infiltration, myeloperoxidase activity, and the expression of intercellular adhesion molecule-1 and vascular cell adhesion molecule 1 were reduced. Thus ***PR*** - ***39*** and PR-11 effectively ***inhibit*** myocardial ischemia-reperfusion injury in the rat in vivo. This effect is mediated by ***inhibition*** of I kappa B alpha degradation and subsequent ***inhibition*** of nuclear factor-kappa B-dependent adhesion molecules. The active sequence is located in the first 11 amino acids, suggesting a potential for oligopeptide therapy as an adjunct to revascularization.

L6 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:936421 CAPLUS
DOCUMENT NUMBER: 136:178301
TITLE: ***PR*** - ***39*** and PR-11 peptides ***inhibit*** ischemia-reperfusion injury by blocking ***proteasome*** -mediated I.kappa.B.alpha. degradation
AUTHOR(S): Bao, Jialin; Sato, Kaori; Li, Min; Gao, Youhe; Abid, Ruhul; Aird, William; Simons, Michael; Post, Mark J.
CORPORATE SOURCE: Angiogenesis Research Center, Beth Israel Deaconess Medical Center, Dartmouth Medical School, Hanover, NH, 03756, USA
SOURCE: American Journal of Physiology (2001), 281(6, Pt. 2),

H2612-H2618
CODEN: A[redacted]AP; ISSN: 0002-9513
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB ***PR*** - ***39*** ***inhibits*** ***proteasome*** -mediated I.kappa.B.alpha. degrdn. and might protect against ischemia-reperfusion injury. The authors studied ***PR*** - ***39*** , its truncated form PR-11, and a mutant PR-11AAA, which lacks the ability to prevent I.kappa.B.alpha. degrdn., in a rat heart ischemia-reperfusion model. After 30 min of ischemia and 24 h of reperfusion, cardiac function, infarct size, neutrophil infiltration, and myeloperoxidase activity were measured. Intramyocardial injection of 10 nmol/kg ***PR*** - ***39*** or PR-11 at the time of reperfusion reduced infarct size by 65% and 57%, resp., which improved blood pressure, left ventricular systolic pressure, and relaxation and contractility compared with vehicle controls 24 h later. Neutrophil infiltration, myeloperoxidase activity, and the expression of intercellular adhesion mol.-1 and vascular cell adhesion mol. 1 were reduced. Thus, ***PR*** - ***39*** and PR-11 effectively ***inhibit*** myocardial ischemia-reperfusion injury in the rat in vivo. This effect is mediated by ***inhibition*** of I.kappa.B.alpha. degrdn. and subsequent ***inhibition*** of nuclear factor-.kappa.B-dependent adhesion mols. The active sequence is located in the first 11 amino acids, suggesting a potential for oligopeptide therapy as an adjunct to revascularization.
REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 4 OF 6 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 2001:935656 SCISEARCH
THE GENUINE ARTICLE: 487UW
TITLE: ***PR*** - ***39*** and PR-11 peptides protect against ischemia-reperfusion injury by ***inhibition*** of ***proteasome*** mediated I kappa B alpha degradation
AUTHOR: Bao J L (Reprint); Gao Y H; Li M; Abid M R; Aird W; Simons M; Post M J
CORPORATE SOURCE: Harvard Univ, Beth Israel Deaconess Med Ctr, Sch Med, Boston, MA 02215 USA
COUNTRY OF AUTHOR: USA
SOURCE: CIRCULATION, (23 OCT 2001) Vol. 104, No. 17, Supp. [S], pp. 52-52. MA 251.
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621 USA.
ISSN: 0009-7322.
DOCUMENT TYPE: Conference; Journal
LANGUAGE: English
REFERENCE COUNT: 0

L6 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:178321 CAPLUS
DOCUMENT NUMBER: 133:205925
TITLE: PR39, a peptide regulator of angiogenesis. [Erratum to document cited in CA132:149677]
AUTHOR(S): Li, Jian; Post, Mark; Volk, Rudiger; Gao, Youhe; Li, Min; Metals, Caroline; Sato, Kaori; Tsai, Jo; Aird, William; Rosenberg, Robert D.; Hampton, Thomas G.; Li, Jianyi; Sellke, Frank; Carmeliet, Peter; Simons, Michael
CORPORATE SOURCE: Angiogenesis Research Center, Department of Surgery, Beth Israel Deaconess Medical Center and Harvard Medical School, Boston, MA, 02215, USA
SOURCE: Nature Medicine (New York) (2000), 6(3), 356
CODEN: NAMEFI; ISSN: 1078-8956
PUBLISHER: Nature America
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The correct versions are given for Figs. 2a, c, and d on page 51; Fig. 3c on page 52; and Fig. 5b on page 53.

L6 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:46162 CAPLUS

DOCUMENT NUMBER: 132:149677
TITLE: PR39, a [redacted]tide regulator of angiogenesis[redacted]
AUTHOR(S): Li, Jian; Post, Mark; Volk, Rudiger; Gao, Youhe; Li, Min; Metais, Caroline; Sato, Kaori; Tsai, Jo; Aird, William; Rosenberg, Robert D.; Hampton, Thomas G.; Li, Jianyi; Sellke, Frank; Carmeliet, Peter; Simons, Michael
CORPORATE SOURCE: Angiogenesis Research Center, Department of Surgery both at Beth Israel Deaconess Medical Center and Harvard Medical School, Boston, MA, 02215, USA
SOURCE: Nat. Med. (N. Y.) (2000), 6(1), 49-55
CODEN: NAMEFI; ISSN: 1078-8956
PUBLISHER: Nature America
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Although tissue injury and inflammation are considered essential for the induction of angiogenesis, the mol. controls of this cascade are mostly unknown. Here we show that a macrophage-derived peptide, PR39, inhibited the ubiquitin-proteasome-dependent degrdn. of hypoxia-inducible factor-1.alpha. protein, resulting in accelerated formation of vascular structures in vitro and increased myocardial vasculature in mice. For the latter, coronary flow studies demonstrated that PR39-induced angiogenesis resulted in the prodn. of functional blood vessels. These findings show that PR39 and related compds. can be used as potent inductors of angiogenesis, and that selective inhibition of hypoxia-inducible factor-1.alpha. degrdn. may underlie the mechanism of inflammation-induced angiogenesis.

REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT 17:57:05 ON 30 MAR 2002

L1 364 S PR-39
L2 21657 S PROTEASOME
L3 9946 S L2 (P) INHIBIT?
L4 364 S L1 (P) L1
L5 9 S L1 (P) L3
L6 6 DUPLICATE REMOVE L5 (3 DUPLICATES REMOVED)

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